FILE 'MEDLINE, EMBASE, BIOSIS, CAPLUS' ENTERED AT 13:05:35 ON 03 MAR 2006
L1 909 S RECA (S) (TRUNCAT? OR DELET?)
L2 46 S L1 (S)((C-TERMIN?) OR (CARBOXY? TERMINAL))
L3 14 DUP REM L2 (32 DUPLICATES REMOVED)
L4 6 S L3 AND PY<=2002
L5 8 S L3 NOT L4

AU Horii T; Ozawa N; Ogawa T; Ogawa H
SO Journal of molecular biology, (1992 Jan 5) Vol. 223, No. 1, pp. 105-14.
Journal code: 2985088R. ISSN: 0022-2836.
TI Inhibitory effects of N- and C-terminal truncated Escherichia coli recA gene products on functions of the wild-type recA gene.
AB The effects of the expression of Escherichia coli truncated RecA protein on the host recA functions were examined. The recA gene on a multicopy plasmid was manipulated to express the truncated RecA protein from its carboxyl (C) and amino (N) terminal ends where a maximum of four extra amino acid residues was added. The regulatory part of the recA gene was substituted by the lacUV5 promoter in the plasmid to facilitate the artificial control of recA expression. Enzyme-linked immunosorbent assay and Western blot analyses revealed great differences in accumulation of the truncated RecA proteins in the cell, depending on the location of the site of truncation. The expression of truncated proteins lacking 62, 77, 93 or 149 amino acid residues from the C-terminal end caused the host recA+ wild-type cell to become sensitive to ultraviolet irradiation and interfered with chromosomal recombination but did not interfere with the induction of lambda prophage. The expression of truncated RecA protein with 25 amino acid residues deleted from the C -terminal end caused the host cell to induce SOS functions constitutively. Truncated RecA proteins with 15 or 28 amino acid residues missing from the N-terminal end severely interfered with all of the host recA functions examined here. The effect of the loss of 41 amino acid residues from the N-terminal end of RecA was significant but less than the effect of proteins lacking 15 or 28 amino acid residues from the N-terminal end. A protein lacking 59 amino acid residues from the N-terminal end showed little interference with any measured recA functions, suggesting that the deletion of the region from around residues 41 to 59, which is rich in hydrophobic side-chains, influenced the ability of the truncated protein to interfere with the functions of wild-type RecA protein. We also constructed a mutant gene with an internal deletion whose product was missing a region from residues 184 to 204. That mutant RecA protein was stably accumulated in the cell. This protein had little effect on the function of host wild-type recA gene product. The possible function of the regions at the N and C termini are discussed.

AU Tateishi S; Horii T; Ogawa T; Ogawa H
SO Journal of molecular biology, (1992 Jan 5) Vol. 223, No. 1, pp. 115-29.
Journal code: 2985088R. ISSN: 0022-2836.
TI C-terminal truncated Escherichia coli RecA protein RecA5327 has enhanced binding affinities to single- and double-stranded DNAs.
AB RecA5327 is a truncated RecA protein that is lacking 25 amino acid residues from the C-terminal end. The expression of RecA5327 protein in the cell resulted in the constitutive induction of SOS functions without damage to the DNA. Purified RecA5327 protein effectively promoted the LexA repressor cleavage reaction and ATP hydrolysis at a lower concentration of single-stranded DNA than that required for wild-type RecA protein. A DNA binding study showed that RecA5327 has about ten times higher affinity for single-stranded DNA than does the wild-type RecA protein. Moreover RecA5327 protein binds stably to double-stranded (ds) DNA in conditions where the wild-type RecA protein could not bind. The binding of RecA5327 protein to dsDNA was associated with the unwinding of dsDNA, suggesting that RecA5327 binds to dsDNA in the same manner as does the wild-type protein. The fact that RecA5327 does not bind stoichiometrically but forms short filaments on dsDNA suggests that it nucleates to dsDNA much more frequently than does the wild-type protein. The role of the 25 C-terminal residues, in the regulation of RecA binding to DNA, is discussed.

AU Marais A; Bove J M; Renaudin J
SO Journal of bacteriology, (1996 Feb) Vol. 178, No. 3, pp. 862-70.
Journal code: 2985120R. ISSN: 0021-9193.
TI Spiroplasma citri virus SpV1-derived cloning vector: deletion formation by illegitimate and homologous recombination in a spiroplasmal host strain which probably lacks a functional recA gene.
AB We have previously described the use of the replicative form (RF) of Spiroplasma citri virus SpV1 as a vector for expressing an epitope of the P1 adhesin protein from Mycoplasma pneumoniae in S. citri (A. Marais, J. M. Bove, S.F. Dallo, J. B. Baseman, and J. Renaudin, J. Bacteriol. 175:2783-2787, 1993). We have now studied the structural instability of the recombinant RF leading to loss of the DNA insert. Analyses of viral clones with deletions have shown that both illegitimate and homologous recombination were involved in deletion formation. For one such clone, deletion has occurred via a double crossing-over exchange between the circular free viral RF and SpV1 viral sequences present in the S. citri host chromosome. The homologous recombination process usually requires the RecA protein. However, characterization of the recA gene of the S. citri R8A2 host strain revealed that over two-thirds of the open reading frame of the recA gene was deleted from the C-terminal part, indicating that this particular strain is probably RecA deficient.

AU Muth G; Frese D; Kleber A; Wohlleben W
SO Molecular & general genetics : MGG, (1997 Jul) Vol. 255, No. 4, pp. 420-8.
Journal code: 0125036. ISSN: 0026-8925.
TI Mutational analysis of the Streptomyces lividans recA gene suggests that only mutants with residual activity remain viable.
AB Temperature-sensitive integration plasmids carrying internal fragments of the Streptomyces lividans TK24 recA gene were constructed and used to inactivate the chromosomal recA gene of S. lividans by gene disruption and gene replacement. Integration of these plasmids resulted in recA mutants expressing C-terminally truncated RecA proteins, as deduced from Southern hybridization experiments. Mutants FRECD2 in which the last 42 amino acids, comprising the variable part of bacterial RecA proteins, had been deleted retained the wild-type phenotype. The S. lividans recA mutant FRECD3 produced a RecA protein lacking 87 amino acids probably including the interfilament contact site. FRECD3 was more sensitive to UV and MMS than the wild-type. Its ability to undergo homologous recombination was impaired, but not completely abolished. Integration of the disruption plasmid pFRECD3 in S. coelicolor "Muller" caused the same mutant phenotype as S. lividans FRECD3. In spite of many attempts no S. lividans recA mutants with deletions of 165 C-terminal amino acids or more were isolated. Furthermore, the recA gene could not be replaced by a kanamycin resistance cassette. These experiments indicate a crucial role of the recA gene in ensuring viability of Streptomyces.

AU Mikoc A; Ahel I; Gamulin V
SO Molecular & general genetics : MGG, (2000 Oct) Vol. 264, No. 3, pp. 227-32.
Journal code: 0125036. ISSN: 0026-8925.
TI Construction and characterization of a Streptomyces rimosus recA mutant: the RecA-deficient strain remains viable.
AB Although previously reported attempts to construct recA null mutants in Streptomyces spp. have been unsuccessful, we have used the suicide plasmid pErmdeltaRecA to inactivate the recA gene in Streptomyces rimosus by gene disruption. pErmdeltaRecA carries the erythromycin resistance gene ermE and a 451-bp fragment of the S. rimosus recA gene (encoding amino acids 2-151). An erythromycin-resistant clone with single plasmid integration into the recA gene on the chromosome was analyzed in detail. This clone possesses one inactive copy of recA which lacks the entire promoter region and the ATG start codon, and a second, truncated gene that encodes only first 151 amino acids of the RecA protein. This S. rimiosus rec A mutant can therefore be considered a completely RecA-deficient strain. The mutant strain is highly sensitive to UV light. Introduction of a plasmid carrying the wild type S. rimosus recA gene completely restored the UV resistance of the recA mutant to wild-type levels. recA genes encoding RecA proteins with short deletions at the C-terminus (21 and 51 amino

acids) could not fully rescue the UV sensitivity of the S. rimosus recA strain, when introduced in the same way.

AU Lusetti, Shelley L.; Shaw, Jeffrey J.; Cox, Michael M.
SO Journal of Biological Chemistry (2003), 278(18), 16381-16388
CODEN: JBCHA3; ISSN: 0021-9258
TI Magnesium Ion-dependent Activation of the RecA Protein Involves the C Terminus
AB Optimal conditions for RecA protein-mediated DNA strand exchange include 6-8 mM Mg2+ in excess of that required to form complexes with ATP. We provide evidence that the free magnesium ion is required to mediate a conformational change in the RecA protein C terminus that activates RecA-mediated DNA strand exchange. In particular, a "closed" (low Mg2+) conformation of a RecA nucleoprotein filament restricts DNA pairing by incoming duplex DNA, although single-stranded overhangs at the ends of a duplex allow limited DNA pairing to occur. The addn. of excess Mg2+ results in an "open" conformation, which can promote efficient DNA pairing and strand exchange regardless of DNA end structure. The removal of 17 amino acid residues at the Escherichia coli RecA C terminus eliminates a measurable requirement for excess Mg2+ and permits efficient DNA pairing and exchange similar to that seen with the wild-type protein at high Mg2+ levels. Thus, the RecA C terminus imposes the need for the high magnesium ion concns. requisite in RecA reactions in vitro. We propose that the C terminus acts as a regulatory switch, modulating the access of double-stranded DNA to the presynaptic filament and thereby inhibiting homologous DNA pairing and strand exchange at low magnesium ion concns.

IN Cox, Michael M.; Lusetti, Shelley L.; Eggler, Aimee L.; Haruta, Nami
SO U.S. Pat. Appl. Publ., 34 pp.
CODEN: USXXCO
TI Protein and cDNA sequences of Escherichia coli enzyme RecA mutants
AB The present invention provides RecA mutant proteins, having either a single mutation or a double mutation. The RecA mutant proteins are highly proficient in both SSB displacement and steady state binding of DNA in the presence or absence of SSB as compared to the wild-type protein. The single RecA mutant, RecA.DELTA.C 17, has 17 amino acid residues removed from the carboxyl terminus. The double mutant RecA, RecA.DELTA.C17/E38K, combines the 17 amino acid residue C -terminal deletion of RecA.DELTA.C17, with a single amino acid change from Glutamate to Lysine at position 38. These RecA mutant proteins are pH sensitive allowing control over formation of products. Hence, methods of using the novel RecA mutants and kits having the RecA mutants as components thereof are also contemplated by the present invention.

AU Eggler Aimee L; Lusetti Shelley L; Cox Michael M
SO The Journal of biological chemistry, (2003 May 2) Vol. 278, No. 18, pp. 16389-96. Electronic Publication: 2003-02-20.
Journal code: 2985121R. ISSN: 0021-9258.
TI The C terminus of the Escherichia coli RecA protein modulates the DNA binding competition with single-stranded DNA-binding protein.
AB The nucleation step of Escherichia coli RecA filament formation on single-stranded DNA (ssDNA) is strongly inhibited by prebound E. coli ssDNA-binding protein (SSB). The capacity of RecA protein to displace SSB is dramatically enhanced in RecA proteins with C-terminal deletions. The displacement of SSB by RecA protein is progressively improved when 6, 13, and 17 C-terminal amino acids are removed from the RecA protein relative to the full-length protein. The C-terminal deletion mutants also more readily displace yeast replication protein A than does the full-length protein. Thus, the RecA protein has an inherent and robust capacity to displace SSB from ssDNA. However, the displacement function is suppressed by the RecA C terminus, providing another example of a RecA activity with C-terminal modulation. RecADeltaC17 also has an enhanced capacity relative to wild-type RecA protein to bind ssDNA containing secondary structure. Added Mg(2+) enhances the ability of wild-type RecA and the RecA C-terminal deletion mutants to compete with SSB and replication protein A. The overall binding of RecADeltaC17 mutant protein to linear ssDNA is increased further by the mutation E38K, previously shown to enhance SSB displacement from ssDNA. The double mutant RecADeltaC17/E38K displaces SSB somewhat better than either individual mutant protein under some conditions and exhibits a higher

steady-state level of binding to linear ssDNA under all conditions.

AU Lusetti Shelley L; Wood Elizabeth A; Fleming Christopher D; Modica Michael J; Korth Joshua; Abbott Lily; Dwyer David W; Roca Alberto I; Inman Ross B; Cox Michael M

SO The Journal of biological chemistry, (2003 May 2) Vol. 278, No. 18, pp. 16372-80. Electronic Publication: 2003-02-20.
Journal code: 2985121R. ISSN: 0021-9258.

TI C-terminal deletions of the Escherichia coli RecA protein. Characterization of in vivo and in vitro effects.

AB A set of C-terminal deletion mutants of the RecA protein of Escherichia coli, progressively removing 6, 13, 17, and 25 amino acid residues, has been generated, expressed, and purified. In vivo, the deletion of 13 to 17 C-terminal residues results in increased sensitivity to mitomycin C. In vitro, the deletions enhance binding to duplex DNA as previously observed. We demonstrate that much of this enhancement involves the deletion of residues between positions 339 and 346. In addition, the C-terminal deletions cause a substantial upward shift in the pH-reaction profile of DNA strand exchange reactions. The C-terminal deletions of more than 13 amino acid residues result in strong inhibition of DNA strand exchange below pH 7, where the wild-type protein promotes a proficient reaction. However, at the same time, the deletion of 13-17 C-terminal residues eliminates the reduction in DNA strand exchange seen with the wild-type protein at pH values between 7.5 and 9. The results suggest the existence of extensive interactions, possibly involving multiple salt bridges, between the C terminus and other parts of the protein. These interactions affect the pK(a) of key groups involved in DNA strand exchange as well as the direct binding of RecA protein to duplex DNA.

EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	227	michael near2 cox.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/03/03 12:46
L2	1	shelley near2 lusetti.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/03/03 12:47
L3	3	aimee near2 eggler.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/03/03 12:47
L4	1	nami near2 haruta.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/03/03 12:46
L5	154	recA with (truncat$3 OR delet$3)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/03/03 12:48
L6	3	l1 and l5	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/03/03 12:48
L7	29	l5 and (("c-termin$3") OR (carboxy adj termin$3))	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/03/03 12:58
L8	1	("5,565,350").PN.	USPAT	OR	OFF	2006/03/03 12:51
L9	24	l5 and (carboxyl adj termin$3)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2006/03/03 12:59